Due largely to the researches of Funk, Vedder, Chamberlain and many others during the last few years, decided progress has been made toward a specific treatment of the disease by the use of appropriate foods and particularly by the use of extract of rice polishings and other substances rich in "vitamines." Some of the reports of the results of this method of treatment have been overly enthusiastic, but definite progress has been made and a sound foundation for further investigation established.

The extremely grave and widespread infantile type of beriberi (taon and many other local names), as well as the multiple neuritis of pregnant and parturient women, constitutes one of the most interesting and baffling problems in tropical neuritis, which may have consideration in a subsequent article.

CLINICAL DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS, ACUTE POLIOMYELITIS, BOTULISM AND CERTAIN FORMS OF FOOD AND DRUG POISONING.

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Epidemic encephalitis, botulism, certain forms of acute poliomyelitis and cerebrospinal syphilis, wood-alcohol poisoning and poisoning by drugs, such as veronal and members of the belladonna group present in common involvement of the cranial nerves resulting frequently in considerable diagnostic difficulty. It is the purpose of this paper to present a comparison of the various points which may be used in differential diagnosis of these conditions.

Poliomyelitis (Acute Poliomyelitis, Polioencephalitis, Infantile Paralysis or Heine-Medin's Disease) was first described by Jörg in 1816, who noted the occurrence of a febrile disease followed by paralysis of the limbs and subsequent club-foot. It is hardly necessary at the present moment to review the contributions to the subject of Heine-Medin, Wickman, Peabody, Draper and Dochez, Flexner and Amoss, Webster, Rosenow and many others more than to say that the old conception of the disease as expressed in the term "infantile paralysis" has given way to the recognition of the fact that the disease is a general infection in which paralysis may or may not occur. It is characterized upon an average by an incubation period of variable duration, usually about a week, followed by a stage of prodromal symptoms which are at times slight and of a very transitory nature and easily overlooked, then an acute stage with paralytic manifestations and lastly a period of retrogression. The prodromal symptoms are of a very general nature and vary from epidemic to epidemic. They may be so slight as to be entirely overlooked, and the first indication of the presence of the disease is then a paralysis. Undoubtedly the most constant and important symptom of this period is fever of an irregular character varying from a slight rise to 103° F. or more. Initial chills are infrequent. Profuse sweating has been noted by Müller in 75 per cent of his cases; other observers failed to notice any sweating in more than 25 per cent. It is evident that this symptom varies considerably. Drowsiness of varying duration and intensity may be present; the patient, however, may be easily aroused, but is usually found to be then irritable to a considerable degree. This irritability manifests itself in an expression of fear of being moved or examined, and is associated with another cardinal symptom, hyperesthesia of the skin and pain on passive motion, specially those motions which cause anterior flexion of the spine—or the legs at the hip-joint and forward bending of the neck. While retraction of the head is not often seen, there is, nevertheless, a tendency of the head to fall back when the child is lifted from the bed by the hands, and there may be found a stiffness of the posterior neck muscles. Test for Kernig's sign results in pain and resistance of the hamstring muscles. These pains and stiffened muscles are a result of meningeal irritation which occurs in the preparalytic stage. Before paralysis takes place there may also be a definite weakness of various groups of muscles, as seen in walking or standing. While convulsions are quite unusual, muscular twitchings are rather frequently seen.

The onset of the disease may be manifested by involvement of the respiratory tract as seen in an inflammatory reaction of the conjunctiva, nose, throat, bronchi or lungs. Regan describes at length the mouth and throat findings in his series of cases, and calls attention to congestion of the throat as an almost constant symptom in the early acute stage accompanied by a varying degree of capillary enlargement on the faucial mucosa and pharynx. The palate, pillars and uvula are a diffused deep red, the soft palate a deep red with distinct purple or violaceous tinge; the hard palate pink—the more severe the deeper the color. In the severe bulbar types in which the prognosis is bad there may be an anemic appearance of the throat. A mild tonsillitis he finds constant. There is never a membrane but spots similar to Koplik's spots sometimes are seen. The tongue is heavily covered with a grayish or white coating, usually moist, and the tip and edges uncovered and slightly redder than normal. The covering is absent in some places, so that the red shows through. The five most important signs of meningeal invasion are: Neck stiffness with weakness of the anterior neck muscles; unwonted drowsiness; hyperesthesia; ataxic gait and sometimes muscle twitchings. The first two combined are almost pathognomonic. In the bulbar type the child may be extraordinarily acute or stuporous. Constipation is usually present and also anorexia. Urinary retention may occur during the fever; diarrhea with green mucous stools may be a predominating feature. Vasomotor disturbances, such as flushing of one ear or cheek, may occur. The spinal fluid usually is abnormal —it may be clear or slightly opalescent, under increased pressure, increased cell content and increased protein. If the clinical picture be considered with the spinal-fluid findings it may be possible to make a diagnosis in the prodromal stage. When paralysis occurs the diagnosis offers less difficulty.

Paralytic Stage. The paralysis may be considered from an anatomic point of view as follows: Spinal paralysis; paralysis of the diaphragm; paralysis of the intercostal muscles; paralysis of the abdominal muscles; paralysis of the neck and back muscles; bulbospinal paralysis and rapidly progressive fatal cases presenting the symptom-complex of Landry's ascending paralysis, in which death ensues from paralysis of the respiratory organs.

The spinal paralyses present no special difficulty in diagnosis; in the great majority of cases muscles of one or both legs are paralyzed and in nearly one-half of the cases the paralysis is limited to the legs. Owing to the fact that the phrenic nerve has a large number of roots originating from the 3d, 4th and 5th cervical segments, paralysis of the diaphragm is comparatively rare, although in fatal cases it is usually present, being preceded, however, by paralysis of the intercostal muscles. Paralysis of the abdominal muscles may take place and is usually difficult of recognition. It is necessary to remember that paralysis of the neck and back muscles may occur. In the former it is impossible for a patient to support the head, which falls helplessly in one direction or another. While this condition may resemble the paralysis seen in botulism, the differentiation, as a rule, presents no difficulties, as in cases of poliomyelitis in which there is paralysis of the neck and back. There is generally, also, extensive paralysis of both arms and legs. The same may be said of most instances of combined spinal and bulbar paralysis which are associated with extensive lesions involving the arms and legs as well as the muscles supplied from the medulla. However, bulbar paralysis may occur without the association of spinal paralysis. The facial nerve is the one most often affected either in whole or in part. Palsies of the ocular muscles, specially the abducens, have been noted, sometimes extensive involvement of the eye muscles and even ophthalmoplegia externa; nystagmus is rare; involvement of the optic nerve practically never occurs; phonation and speech may be interfered with and swallowing is sometimes difficult. The tongue may be protruded to one side, or there may be bilateral involvement with inability to protrude the tongue at all. When bulbar paralyses occur in conjunction with spinal paralyses they are much less apt to be severe or lasting than when they occur alone. Associated with these high lesions is an ataxia of the arms and legs with exaggeration of the knee-jerks and Achilles reflexes and a tendency to stiffness of the legs. Romberg's sign may be markedly present. While the exaggeration of the reflexes may be persistent for a long time the ataxia rapidly disappears.

In the rapidly progressive fatal cases the picture of an ascending paralysis is presented. The mind is usually clear and the patient dies when the diaphragm and the intercostal muscles become involved. Just as the occurrence of paralysis of an arm or leg may be a very sudden manifestation, so also may a sufficient paralysis to cause death occur in a very short time, sometimes within five hours. Ordinarily, however, there is at first a more or less extensive paralysis which involves either the diaphragm or the intercostal muscles followed by an interval of several days, then a slow extension of the paralysis until respiration finally ceases. Fever is always present in the fatal cases, ranging, as a rule, from 101.5° F. to 102.5° F., and sometimes not over 100° F. This occurrence of fever with the onset of the disease is in sharp contradistinction to the subnormal temperature which occurs in the early stages of botulism, fever not being present until a complication such as bronchopneumonia sets in. In both of these conditions, however, the mind is remarkably clear, differing from that seen in encephalitis lethargica. In the bulbar paralyses of poliomyelitis the mouth secretions are not diminished as in botulism, and a frothy saliva fills the mouth and collects between the lips, while in botulism the salivary secretion is diminished, as are all other secretions, and a thick viscid mucous difficult of expulsion fills the throat.

Cerebral Type. Strümpell, in 1885, suggested the name polioencephalitis for a group of cases of cerebral paralysis in children, and pointed out the resemblance to poliomyelitis. The onset of the disease was sudden with a febrile initial phase, vomiting, convulsions and prodromal period, sometimes absent, lasting for two or three days, sometimes over a week, marked by rapid convulsive seizures; after this time the child was found to have a hemiplegia which gradually began to improve. The paralysis was of the upper segment type without atrophy or reaction of degeneration and with exaggerated reflexes. There remained afterward in some of his cases permanent disturbances of cerebral function such as athetosis, epileptic seizures, speech disturbances and diminution of intelligence. He pointed out that, as in poliomyelitis, the lesion was in the gray matter of the cerebrum, the cortex. The final proof that typical cerebral paralyses have the same etiology as poliomyelitis, in which the transference of the virus to monkeys and the causing in them of poliomyelitis has not been accomplished, but both a flaccid and a spastic paralysis have been found in the same patient and a flaccid paralysis in one member of a family and a spastic paralysis in other members of the same family.

Abortive Types. This is the term applied by Wickman to those cases which show the usual prodromal or preparalytic symptoms, but which do not develop paralysis. These may be cases which run the course of a general infection in which meningeal irritation is specially marked or in which pain is a very marked feature, as in influenza or cases with predominating gastrointestinal disturbances. The serum of these patients acts in the same way that the serum of those who develop paralysis does. It neutralizes the virus *in vitro,* and it is interesting to observe that the serum of a patient who had been paralyzed thirty years before was still able to protect a monkey from the virus of poliomyelitis. Peabody, Draper and Dochez in their monograph point out that it is quite probable that in the past many of the abortive cases have been unrecognized, and in some cases at least the apparent immunity of adults may be due to some previous unrecognized attack.

Epidemic Encephalitis. Epidemic encephalitis, like acute poliomyelitis, is a contagious disease due to a filtrable virus that exists in the nasopharyngeal secretion and in which the nasopharynx serves as a portal of acquisition and dissemination of the disease. While the virus may be preserved for a long period of time in glycerin its virulence is diminished by drying, and while undoubtedly it is usually a droplet-borne disease and propagated directly by the exhalations of the mouth from one person to another, it is, nevertheless, possible that the disease may be carried by insects, such as flies, as is also true of poliomyelitis. Like poliomyelitis also the possibility of convalescent or immune carriers must be considered as a possible and a probable source of the disease. The pathology consists typically of microscopic perivascular lymphocytic infiltrations scattered through the basal ganglia and particularly marked in the brainstem; small hemorrhages may occur, while visceral manifestations have been observed, such as enlargement of the spleen and mesenteric glands and ecchymoses in the pleura, pericardium, bladder and stomach. Visceral changes at autopsy are very inconstant. The blood examination shows nothing characteristic, although a moderate leukocytosis is the rule. The spinal fluid shows little or no change, the fluid is clear; the pressure is not increased; there may or may not be a slight increase of cells (lymphocytes); globulin is usually increased; Lange's colloidal gold test varies. According to Moffitt it may be entirely negative, quite frequently moderately luetic and more rarely an outspoken paretic one. He remarked that the Wassermann reaction is uniformally negative except in complicating cerebrospinal syphilis.

A characteristic feature of epidemic encephalitis is the spread-out course of the occurrence of the symptoms. This is in sharp contradistinction to the rule in poliomyelitis, in which the entire extent of the paralysis may be accomplished in a few hours or at most in a few days. In one of my cases after an abrupt onset in which there was sudden double vision, gastric disturbances, horizontal and vertical nystagmus, unilateral ptosis, optic neuritis, inability to stand, lasting an hour or two, the patient improved during the course of a week, when a renewal of the symptoms occurred with paralysis of the facial nerve that in its turn disappeared in six or eight days, when suddenly an involvement of the hypoglossal took place with marked deviation of the tongue. One of Moffitt's cases died thirteen months after the onset from a relapse of the same type of the disease she had had at first. Others of his died from twelve days to seven weeks after the onset.

If one remembers the typical lesions of epidemic encephalitis, the small areas of lymphocytic infiltration lying about the blood-vessels and that they may occur in any part of the nervous system, he may more readily understand the varying combinations of symptoms, and if it be assumed that the virus of the disease is active within the patient for a varying period of time, sometimes for many weeks, the cause of the peculiar progression of the occurrence of symptoms will be evident.

If two or more of the cardinal symptoms of the disease occur—that is, a state of lethargy or somnolence, marked asthenia, fever of irregular type and cranial nerve palsies—epidemic encephalitis should be suspected. The suspicion is strengthened if the condition has been preceded by influenza. Fever may be slight and be entirely overlooked. The palsies most frequently involve the ocular muscles; there may be ptosis, more or less complete, strabismus, lateral and vertical nystagmus, pupillary changes, occasionally optic neuritis, all of which may be either unilateral or bilateral. Facial palsies occur, sometimes involving a muscle or small group of muscles, sometimes giving a Parkinsonian mask-like expression to the entire face. Pain, myoclonus and convulsions may occur and meningeal symptoms have been noted as dominating the picture. The pains may be of extreme severity, sharp, localized and extremely intractable; these pains may occur in any part of the body, as a finger, ear, in the abdomen, the teeth, the eye or elsewhere. They frequently precede myoclonus and usually stop when the myoclonic jerking of the muscles begins. Convulsions due undoubtedly to cortical irritation may be the initial symptom or may occur later in the disease. In mild cases somnolence may not be very marked, but, as a rule, lethargy distinguishes it from the mental condition of poliomyelitis or botulism. A varying degree of optic neuritis further distinguishes it from these two conditions.

Botulism. Botulism is a state of intoxication which follows the ingestion of the toxins produced by Bacillus botulinus, either type A or B. It can be mistaken for methanol poisoning, epidemic encephalitis and cerebrospinal syphilis. Its symptoms usually occur from twelve to twenty-four hours after eating food containing the botulinus toxin, and may be preceded by gastrointestinal disturbances, particularly in the more severe and fatal cases. Symptoms may occur as early as two hours after or may be delayed for two or three days or even longer in making their appearance. One case has been reported nine days after and another as late as fourteen days, but these are indeed unusual.

The symptoms are classified as follows:

1. Arrest of secretions or hypersecretions of saliva and mucous secretions of the mouth and pharynx.
2. Ophthalmoplegia, externa and interna, more or less complete (blepharoptosis, mydriasis paralysis of accommodation, diplopia, strabismus).
3. Dysphagia, aphonia and rebellious constipation, urinary retention.
4. General weakness of all voluntary muscles.
5. Absence of fever and disturbance of sensation and absence of impairment of intelligence.
6. Respiratory difficulties and circulatory troubles, which frequently result fatally more or less rapidly from bulbar paralysis. There is more or less incoördination of muscular movements of the arms and legs, difficulty in picking up small objects, ataxic gait, usually no abnormalities of tendon reflexes.

The patient complains chiefly of double vision, of weakness, of difficulty in walking. There is pronounced difficulty in swallowing and in the expulsion of the thick, viscid, glairy mucus which accumulates in the throat. Agonizing strangling spells resulting in marked cyanosis are of frequent occurrence and often cause death, due to involvement of the respiratory muscles, either intercostals or the diaphragm, or both. There may be a sensation of great weight or pain in the chest and marked difficulty in breathing. The constipation is due to paralysis of the muscles of the wall of the intestines and may resist all efforts to overcome it for quite a number of days. The diagnosis, especially in sporadic cases, may be a matter of considerable difficulty. It may be confused with acute poliomyelitis, but there is an absence of prodromal symptoms, absence of fever, and in by far the greater number of instances of poliomyelitis there occurs definite paralyses of groups of muscles in the extremities. The posterior muscles of the neck in botulism, or indeed all of the muscles of the neck, may be paralyzed, so that the head has a tendency to fall forward. The patient frequently uses his hands to move his head. In poliomyelitis there is a stiffness of the muscles of the back of the neck and a weakness of the anterior muscles, with a tendency for the head to fall backward. There is also a varying degree of pain on movement in poliomyelitis. In poliomyelitis the mouth secretions are not diminished. The pupils are widely dilated in botulism.

In differentiating botulism from cerebral spinal syphilis the difficulties may be very great, but manifestations of the latter are so generally well known that it is not deemed necessary to more than call attention to the resemblance of the two conditions.

Poisoning by drugs of the belladonna group (belladonna, stramonium, hyoscyamus) show inhibition of secretions and dilation of pupils as occurs in botulism. In belladonna poisoning mild cerebral symptoms regularly occur. The face and neck are flushed either uniformly or in blotches; there is an erythematous eruption of the throat and sometimes generally over the skin resembling that in scarlet fever; the respirations are rapid and deep; the arterial pressure is above normal and the patients pass into a chattering delirium with confusion or even into a maniacal condition; there may be a rise of several degrees of fever. Concentrated urine dropped into a cat's eye will dilate the pupil. In the final stages the heart fails, the blood-pressure is low and death takes place from failure of respiration.

Curare, a South American arrow poison, produces death by respiratory paralysis, as does also conium or poison hemlock. Socrates noted while dying of this poison that there was a paralysis with slight numbness beginning in the toes and gradually ascending until it involved the trunk. Gelsemium acts peripherally like conium, but is more depressing centrally. It has an atropine-like action on the pupil and accommodation.

Veronal Poisoning. I have seen two patients undoubtedly suffering from veronal poisoning, one of which resembled encephalitis. She showed after the ingesting of 105 grains of veronal over a course of four days marked double ptosis, double vision, pronounced lateral nystagmus (except when the eyes were at rest), negative pupils, normal reaction to light and distance, no paralysis of cranial or other nerves, no retinal changes, no gastrointestinal disturbance, no diminution of secretion.

Methanol or Wood Alcohol Poisoning. Since the advent of prohibition there has arisen a formidable mass of literature upon this subject. In acute methanol poisoning there occurs: (1) Unconsciousness terminating either in death or recovery of mentality before twenty-four to forty-eight hours; (2) cyanosis; (3) pupils equal and enlarged, sometimes widely dilated with sluggish reaction to light; (4) respirations slower than normal, ten to twenty per minute; temperature, 98.6° to 100° F. Chemical analysis of early catheterized urine may show presence of formaldehyde; the stomach content may contain methanol. The most striking evidence of this poisoning is blindness, due to optic neuritis followed by atrophy, which has been known to occur after the ingestion of so small a quantity as a teaspoonful or two.

Acute methanol poisoning may present: (1) A mild intoxication with some dizziness, nausea and mild gastrointestinal disturbance followed in a few days by recovery, but occasionally resulting in more or less serious damage to vision; (2) more pronounced effect with marked dizziness, outstanding persistent nausea, more or less severe gastroenteritis, dimness of vision frequently increasing to blindness; (3) overwhelming prostration with coma and death. The patient invariably dies in an unconscious condition, or having regained consciousness suffers relapse and death follows shortly. Generally will be witnessed in severe methanol poisoning what appears to be the ordinary effects of alcoholism—vertigo, nausea, cloudiness of mental functions, exaltation of ideas, general malaise, muscular incoördination and disturbance of vision; blindness may take place very suddenly. The condition of the optic nerve and the mental condition should clearly differentiate it from botulism and poliomyelitis.

Poison from decomposed fish and shell fish bears resemblance to botulism, but the symptoms of poisoning follow more quickly after ingestion of food, is marked by predominating gastrointestinal disturbances and the well-known skin manifestations, specially urticaria.

Conclusive proof of the diagnosis of botulism rests in the recognition of the presence of the toxin of bacillus botulinus or the discovery of the organism itself, which is always present in the contaminated food. The suspicious food and the containers of it should be carefully searched for and subjected to bacteriologic tests such as described by Meyer. Inasmuch as the toxin of type A, which commonly gives trouble in the United States, is poisonous to chickens, Graham has suggested the feeding of suspected material to chickens, which will show characteristic manifestations of botulism. It should be remembered that the ingested food may remain for many days in the stomach and in the intestines of the patient. This fact should be taken advantage of in examining the intestinal content of the patient as well as the stomach content for bacillus botulinus and for its toxin.

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